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International Consensus Report on the Diagnosis and Management of Rhinitis

International Rhinitis Management Working Group

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Contents

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Preface

Consensus, *n. agreement (of opinion) or of different organs effecting purpose*

– Shorter Oxford Dictionary

The nose as gatekeeper of the respiratory tract is continually required to react to environmental change and to rebuff external assault from a variety of agents. Exaggeration or perversion of this defensive response produces the familiar symptoms of rhinitis. With evidence of an increasing prevalence of the disease in its many forms, it seemed an appropriate moment to develop guidelines on the management of the condition. To this end, a working party of leading clinicians and researchers in the fields of allergy and otorhinolaryngology, representing ten countries, were brought together to consider underlying mechanisms and to develop an international consensus statement on the diagnosis and management of rhinitis.

Members of the International Rhinitis Management Working Group met three times during 1993 and the document has been reviewed by other experts in the field. It is intended principally for use by the primary care physician but it is also hoped that it encompasses information and an approach which will be of interest to medical and surgical rhinologists alike.

I have been most fortunate in having such a distinguished group of participants and I would like to thank them for their hard work and for meeting the stringent deadlines which I set. This consensus report represents a distillation of current opinion which takes into account the philosophical differences which exist around the world. It is our hope not only that it provides practical advice but that it will also act as a stimulus to future discussion and research.

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Definition and classification of rhinitis

Definition

Rhinitis is defined as inflammation of the lining of the nose, characterised by one or more of the following symptoms: nasal congestion, rhinorrhoea, sneezing and itching.

Classification

1. **ALLERGIC**
   - SEASONAL
   - PERENNIAL

2. **INFECTIOUS**
   - ACUTE
   - CHRONIC
     - Specific
     - Non-specific

3. **OTHER**
   - IDIOPATHIC
   - NARES
   - OCCUPATIONAL
   - HORMONAL
   - DRUG-INDUCED
   - IRRITANTS
   - FOOD
   - EMOTIONAL
   - ATROPHIC

DIFFERENTIAL DIAGNOSIS

POLYPS

MECHANICAL FACTORS
- DEVIATED SEPTUM
- HYPERTROPHIC TURBINATES
- ADENOIDAL HYPERTROPHY
- ANATOMICAL VARIANTS IN THE OSTIOMEATAL COMPLEX
- FOREIGN BODIES
- CHOANAL ATRESIA

TUMOURS
- BENIGN
- MALIGNANT

GRANULOMAS
- WEGENER'S GRANULOMATOSIS
- SARCOID
- INFECTIOUS
  - Tuberculosis
  - Leprosy
- MALIGNANT – MIDLINE DESTRUCTIVE GRANULOMA

CEREBROSPINAL RHINORRHOEA

Epidemiology of rhinitis

Rhinitis is a very common disease but surprisingly little is known about its epidemiology, probably due to the fact that diagnosis relies on recognition of a symptom complex of varying severity. Surveys of normal subjects not considered to suffer from rhinitis have shown that nevertheless 40% had experienced nasal symptoms the previous day (Sibbald, 1993). There may be a continuum in the frequency and severity of nasal symptoms from subjects with no detectable illness to those with severe disease. Changing patient behaviour, diagnostic fashion and research methods may explain much of the variation in the prevalence of rhinitis between populations and over time.

The prevalence of diagnosed hay fever amongst patients attending general practitioners is 11 per thousand in Denmark, 20 per thousand in England and Wales and 86 per thousand in Australia. These figures underestimate the frequency of the disease since they exclude those not seeking medical help and those in whom rhinitis is not recognised by the physician. Community-based studies provide more accurate information. In one study in London of adults between the ages 16 and 65 years the minimum prevalence of rhinitis was 16%: of these 8% had perennial symptoms, 6% both perennial and seasonal symptoms and 2% seasonal symptoms alone (Sibbald & Rink, 1991).

The peak prevalence of hay fever occurs between 5 and 15 years in England and Wales, between 10 and 19 years in Denmark and at age 24 in the United States, thus affecting 15–20% of students (Broder et al., 1974). Males outnumber females in childhood but the sex ratio becomes even in adulthood. Worldwide, the prevalence of hay fever in school age children is lower in European countries than in the USA or Australia, probably as the result of the allergenicity of the prevailing pollen and aeroallergen burden.

Hay fever appears to be more common in urban than rural areas. In Denmark the prevalence of hay fever amongst patients attending general practitioners was 19% in Copenhagen and 6–11% in rural areas (Pedersen & Weeke, 1981). In the USA, a community study indicated that in those individuals with allergic rhinitis, 75% resided inside the city, compared to 25% who lived in the surrounding rural area (Broder et al., 1974). The reasons for these differences are not known, but may include air pollution. Other factors which may positively influence the development of seasonal allergic rhinitis include order of birth, birth during the pollen season and the presence of a family history of allergy. Hay fever is commoner in non-whites than whites and in upper than lower social classes (Sibbald, 1993) though some of these statistics may be based on greater awareness rather than a greater prevalence.

Hay fever is probably becoming more common. Medical examination of Swedish army recruits has shown that the prevalence of hay fever increased from 4.4% in 1971 to 8.4% in 1981 with a higher increase in the more northerly region of the country; this developing geographical difference argues against the increase being attributable to changes in doctors' labelling preference (Åberg, 1989). The prevalence of allergic skin test reactivity, i.e. atopy, increased from 39 to 50% in a community sample in the USA of individuals of all ages followed for a mean of 8 years (Barbee et al., 1987). The association of skin reactivity with allergic disease suggests that if atopy is increasing, so may allergic diseases such as hay fever.

Mechanisms of rhinitis

Most information regarding the mechanisms of rhinitis concerns allergy and few studies have focused on non-allergic aspects.

Immediate allergic nasal symptoms of **itching, sneezing and watery rhinorrhoea** occur as a consequence of the IgE-dependent activation of mast cells in the nasal mucosa. Mediators released may be preformed and granule-derived (for example, histamine, tryptase). Newly formed, membrane-derived mediators include leukotrienes (LTB_4 and LTC_4) and prostaglandins (PGD_2). A further lipid-derived mediator is platelet-activating factor (PAF). The biological properties of these mediators include vasodilatation and an increase in vascular permeability which may cause **nasal blockage**. Increased glandular secretion results in **mucous rhinorrhoea.** Stimulation of afferent nerves may provoke **itching and sneezing**. Afferent nerve stimulation by mediators (particularly histamine) may also promote an axon reflex with local release of neuropeptides (substance P, tachykinins) which have the potential to provoke further mast cell degranulation. An hypothesis relating to the mechanism of allergic rhinitis is summarised in Fig. 1.

A characteristic feature of allergic inflammation is the local accumulation of inflammatory cells, including CD4+ T lymphocytes, eosinophils, basophils and neutrophils. Eosinophils release a number of highly positively charged basic proteins which may be toxic to human respiratory epithelium and, in the presence of halide ions, may also promote further mast cell degranulation. Unlike neutrophils, the predominant leukotriene released from eosinophils is LTC_4, which may promote mucous rhinorrhoea and nasal congestion. The mechanism of this local tissue eosinophilia is not clear. Possible mechanisms include increased chemotaxis, increased vascular adhesion or possibly enhanced survival of eosinophils in tissues. Recent evidence suggests that a number of peptide mediators (cytokines) may be responsible. Originally described as products of T lymphocytes, cytokines may be released from alternative cells including mast cells, basophils, macrophages and epithelial cells. Interleukin-4

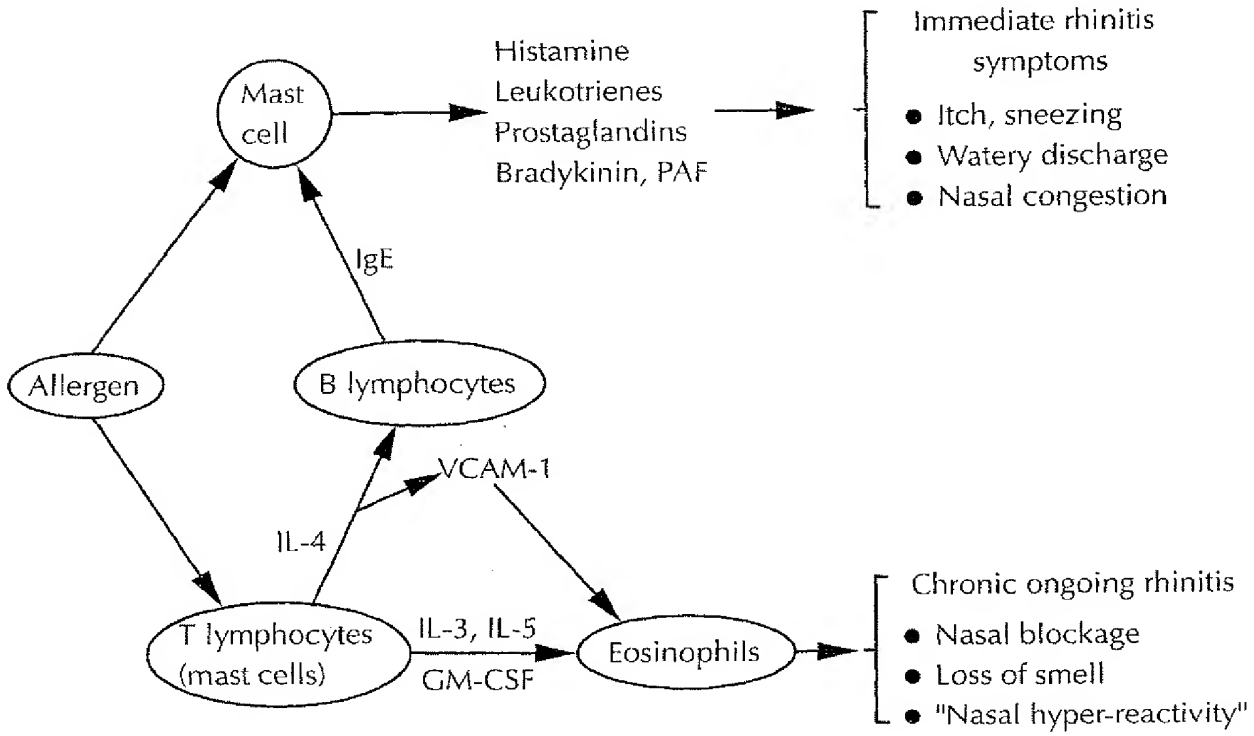


Fig. 1. Hypothesis on mechanisms of allergic rhinitis (reproduced with kind permission of S.R. Durham).

(IL-4) promotes B cell isotype-switching in favour of IgE. In addition IL-4, by promoting increased expression of adhesion molecules (VCAM-1) on vascular endothelium may also selectively recruit eosinophils (Fig. 1). IL-3 stimulates mast cell maturation. IL-5 has properties selective for eosinophils which include the differentiation and maturation of eosinophils from bone marrow precursors, activation of eosinophils for mediator release and enhanced eosinophil survival in tissues.

What then is the evidence for involvement of mediators and inflammatory cells in allergic responses? Mediator concentrations and cells have been measured in nasal lavage fluid and cell infiltration in the nasal mucosa has been assessed in biopsies obtained following local allergen provocation and during natural allergen exposure.

Allergen challenge

Following nasal allergen challenge immediate symptoms of **itching, sneezing and rhinorrhoea** occur within minutes. Depending on the allergen dose administered and the allergen sensitivity of the individual, a proportion of allergic subjects go on to develop a late phase response at 6–12 hours. The development of late responses results in an increase in nasal hyperresponsiveness which may be specific (for the allergen, referred to as 'priming') or non-specific (referring to a heightened sensitivity to irritant triggers such as tobacco smoke or domestic aerosol sprays). Recent studies of nasal lavage have confirmed increases in mediators of hypersensitivity during both early and late nasal response (Naclerio et al., 1985). Cell counts in nasal lavage have confirmed an increase in eosinophils, particularly during late phase responses (Bascom et al., 1988a). Increases in basophils are also observed (Bascom et al., 1988b). More recent studies involving nasal mucosal biopsy have confirmed an increase in CD4+ T lymphocytes as well as neutrophils and eosinophils during late responses (Varney et al., 1992). In situ hybridisation studies using gene probes directed against specific cytokines have confirmed an increase in messenger RNA expression particularly for IL-4 and IL-5 (Durham et al., 1992). It is of interest that the level of mRNA expression of these so-called 'Th2-type' cytokines with known eosinophil-modifying properties was closely correlated with the allergen-induced local increases in eosinophil numbers in the nasal mucosa during late responses.

Nasal priming

When repeated nasal provocations are performed, the number of pollen grains required to elicit a positive response is markedly reduced (Connell, 1969). This priming effect disappears when the interval between challenges is increased beyond one week. This 'priming' of the nasal mucosa is thought to be due to the influx of inflammatory cells attracted to the mucosa following the initial challenge (Bentley, 1992). The clinical importance of this phenomenon is that patients allergic to tree pollens may be 'primed' during the springtime and develop marked grass pollen-induced symptoms even following exposure to very low pollen counts. This emphasises the importance of early intervention with anti-inflammatory treatment, preferably before exposure begins.

Seasonal exposure

Studies of cells infiltrating the nasal mucosa during the pollen season have confirmed an increase in eosinophils. More importantly, the number of 'activated' secreting eosinophils is increased during seasonal exposure. Epithelial migration of mast cells is also observed (Viegas et al., 1987; Bentley et al., 1992). Inflammation persists throughout the pollen season and results in nasal hyper-reactivity characterised by exacerbation of nasal symptoms when the patient is exposed to non-specific or irritant triggers such as tobacco smoke, strong or noxious smells, changes in temperature or exercise.

Perennial allergic rhinitis

Chronic inflammation of the nasal epithelium and submucosa is the cardinal feature of patients with chronic symptoms which occur as a consequence of exposure to perennial allergens such as the dust mites (*Dermatophagoides pteronyssinus* and *D. farinae*), cockroaches and mammals, particularly pets such as cats and dogs, and horses. Inflammation is composed of mast cells, eosinophils, T lymphocytes and macrophages. Its persistence explains why prolonged regular treatment with topical corticosteroids is frequently required to control symptoms. Nasal biopsies from patients with perennial rhinitis have confirmed the above features, a major point being the intensity and persistence of the allergic inflammatory response (Bradding et al., 1992).

Non-allergic rhinitis

The term "non-allergic rhinitis" is reserved for inflammation of the nasal mucous membrane which is unrelated to either allergy, infection, structural lesions or other systemic diseases (Togias, 1993). It is therefore a diagnosis of exclusion. The previously used term 'vasomotor rhinitis' implies that the cause is known. This is certainly not the case and the term is probably therefore best replaced by 'idiopathic rhini-

tis'. Patients within this classification typically complain of symptoms of **sneezing, watery rhinorrhoea or nasal obstruction** following exposure to irritants or, in particular, changes in temperature. Their symptoms may be reproduced by challenge with cold dry air. Recent studies have indicated that cold dry air-induced rhinitis may also occur as a consequence of the release of mast cell associated mediators involving a non-IgE-dependent mechanism. It is possible that mast cell activation occurs as a consequence of changes in osmolality of nasal secretions following provocation. Neurogenic mechanisms may also be important. For example, patients who develop excessive watery rhinorrhoea after exposure to temperature changes or after eating spicy food may gain relief from use of topical anticholinergic drugs such as ipratropium bromide (Dolovich et al., 1989). A further 'non-allergic' group of rhinitis sufferers include those subjects sensitive to aspirin. This may be accompanied by severe rhinitis symptoms with or without associated nasal polyps and asthma. One hypothesis is that by blocking the cyclo-oxygenase pathway, aspirin shifts arachidonic acid metabolism towards lipoxygenase with excess generation of leukotrienes (Christie et al., 1991) which may promote immediate nasal symptoms of mucus, rhinorrhoea and nasal obstruction.

Quality-of-life issues in rhinitis

Patients with rhinitis are not just troubled by nasal symptoms. The condition impacts heavily on health-related quality of life. Patients are limited in their inability to do everyday activities, concentration is impaired, associated symptoms such as headache are troublesome, practical things such as remembering to carry a handkerchief and repeatedly blowing the nose are a nuisance, sleep is impaired, social interaction is limited and there is an impact on emotional well-being (Juniper & Guyatt, 1991; Juniper et al., 1993; Bousquet et al., in press).

Although the direct societal cost of rhinitis may not be high, as few patients require hospitalisation or extensive use of health services, the economic impact on patients themselves may be considerable. Loss of earnings may not only be attributable to sick leave, but also occurs due to impaired concentration and reduced productivity. Personal income is spent on items such as home/car air conditioners and air filters. For those sensitive to house dust mite, home refurnishing can be expensive and for pollen sensitive patients, holiday venues and accommodation may add to the financial burden. In addition, the cost of medication may not always be available through national health or personal insurance schemes.

Types of rhinitis

Seasonal allergic rhinitis

Symptoms

The main symptoms are itching and irritation in the nose, sneezing and watery rhinorrhoea, often associated with nasal congestion. Allergic rhinitis may be accompanied by itching in the throat, eyes and ears, epiphora and oedema around the eyes. Around 20% of cases are accompanied by symptoms of asthma (Smith, 1983). Allergic rhinitis is at best a nuisance and at worst can be incapacitating. It may be complicated by headache, fatigue and lack of concentration. With increasing age, symptoms generally decline but can persist or appear even in old age.

Seasonal allergens

Pollens responsible for seasonal allergic rhinitis are wind-borne tree pollen in the spring, such as alder, hazel, oak, elm and birch; grass pollen in early to mid-summer, mainly timothy, ryegrass and, in the southern states of America, Bermuda grass; and weed pollen in late summer to frost, mugwort, ragweed and plantain. In non-tropical parts of the world, seasonal allergic rhinitis occurs in defined periods of the year. The type of allergen is geographically determined by the climate in that particular area but it should be remembered that pollen allergens can be transported by the wind considerable distances, producing 'out of season' symptoms.

In temperate climates of the Northern hemisphere, the pollen season may start in February and March with alder and hazel. In May birch (*Betula*) pollen is the most important allergen; in June and July grass pollens prevail. In the autumn, weed pollens such as mugwort pollinate. In eastern Europe and the eastern part of North America, ragweed (*Ambrosia*) predominates in the late summer and early autumn. In southern Europe, the pollen seasons begin earlier, are more protracted and involve tree pollen such as cypress and the olive tree, and the wheat plant.

Mould spores in the outdoor air have a seasonal variation generally with reduced numbers during the winter months and sufficient numbers to cause allergic symptoms during the summer and autumn months (e.g., *Cladosporium, Alternaria*) (Platts-Mills et al., 1987). It is possible that mould allergy preferentially gives rise to bronchial asthma and a better correlation has been found between mould spore counts and symptom score in ragweed allergic patients compared to pollen counts. This may partly be explained by the smaller size of mould spores compared to pollen grains. Although house dust mite is usually regarded as a perennial allergen, there may be an increased number of house dust mites in the damp autumn months. Such local conditions can form the basis for specific sensitization and seasonal outbreaks of allergic symptoms. In northern Sudan outbreaks of asthma and rhinitis can occur during the dry winter months due to small midges, *Cladotanytarsus lewisi*, or the green nimitii midge, which reproduce in stagnant water when the rivers are low (Kay et al., 1983). It is, therefore, important to know about such local factors in diagnosing seasonal allergic rhinitis.

Pollen counts

Registration of pollen and spore content of the air has been performed in the USA from 1916 and in the UK from 1942, after which many countries have followed. The pollen counts are of interest in evaluation of clinical trials in order to relate symptoms to pollen counts and daily pollen counts available through the media are a source of interest to patients. The counts may be of use in forecasting the start and intensity of the pollen season though factors such as expected rainfall, wind speed, hours of sunshine, temperature and cloud height may affect the accuracy of such predictions. In the south of England a relationship has been found between the mean temperature for April and May and the intensity of the following grass pollen season (Davies & Smith, 1973).

In studies of allergic seasonal rhinitis, a correlation between the daily pollen count and overall daily symptom score and medication score has been found. The symptoms on any particular day will be influenced by exposure on that day but also on previous days due to the priming phenomenon. As a consequence, at the end of the pollen season, it is usual to observe a decline in symptoms which is slower than that of the pollen counts themselves (Brostrom & Moller, 1990). Individual sensitivity will also influence the intensity of symptomatology. In highly sensitive individuals, many symptoms occur with pollen counts of 15–75 pollen grains/m^3 per 24 hours, whereas in the less sensitive, 4–10 times this exposure may be necessary to provoke equivalent symptoms (Taudorf & Mosholm, 1988).

Cross reactions in pollen allergy

Pollen grains contain species-specific allergenic substances as well as allergens with similarities between genera and families. As a consequence, a patient may manifest immunological cross reactivity due to IgE

antibody production in response to structurally related allergens. Cross reactivity can also occur between pollen and food allergens. Allergy to birch is often associated with oral allergy syndrome, characterised by itching and occasionally oedema on eating fresh fruit such as apple, peach and cherry, or carrots and hazelnuts (Ortolani et al., 1988). Cross reactivity also exists between mugwort pollen and certain spices or celery and between ragweed and melon and banana.

Perennial allergic rhinitis

Indoor allergens are usually present on a perennial basis; most important among these are house dust mites and animal danders, with cockroaches and certain mould species being relevant in some areas (Jacobs, 1987). The symptoms of perennial allergic rhinitis are the same as those of seasonal allergic rhinitis, but nasal blockage is usually more pronounced and eye itching is rarely a problem. The symptoms are chronic and persistent and patients may present with secondary complaints of mouth-breathing, snoring, sinusitis or 'a permanent cold' (Lucente, 1989).

Infectious rhinitis

Infectious rhinitis may be acute or chronic. **Acute infectious rhinitis** is usually due to a large range of viral agents though there may be secondary bacterial infection with sinus involvement. The commonest bacterial pathogens are *Streptococcus pneumoniae* and *Haemophilus influenzae* (Axelsson & Brorson, 1972; Goldman, 1987; Gwaltney et al., 1992).

Chronic infectious rhinitis may be caused by a specific organism as in tuberculosis (*Mycobacterium tuberculosis*), rhinoscleroma (*Klebsiella rhinoscleromatis*), leprosy (*M. leprae*), syphilis (*Treponema pallidum*), yaws (*T. pertenue*) or glanders (*Loefflerella mallei*). A considerable number of fungal agents may also produce infection in the nose and sinuses, the commonest of which is the *Aspergillus* genus. Even protozoan infection (leishmaniasis) and parasites such as the *Chrysomyia* fly may be responsible for infection in the nose (Weir, 1987).

When symptoms persist, with concomitant sinusitis a chronic situation is deemed to exist after eight to twelve weeks. The symptoms of **chronic infectious rhinosinusitis** are nasal congestion, nasal discharge which is predominantly mucopurulent, facial pain and pressure and olfactory disturbance.

Allergy, mucociliary disturbance and immune deficiency may predispose certain individuals to the development of chronic infection (Mackay & Cole, 1987; Lund & Scadding, 1991). Mucociliary abnormalities may be congenital, as in primary ciliary dyskinesia (Afzelius, 1976; Pedersen & Mygind, 1976), Young's syndrome (Young, 1970) or cystic fibrosis, or secondary to infection. Similarly immune deficiency may be congenital or acquired.

Other types of rhinitis

Non-allergic non-infective rhinitis comprises a heterogeneous group of patients. **Idiopathic rhinitis** is probably a better term than 'vasomotor rhinitis' as these patients present with 'nasal hyperresponsiveness' to non-specific triggers such as strong smells (perfumes, bleach, solvents), irritants such as tobacco smoke, dusts, exhaust fumes and changes in environmental temperature and humidity. The exact mechanism is unknown.

A subgroup, **non-allergic rhinitis with eosinophilia syndrome (NARES)** (Jacobs, 1987) is characterized by the presence of nasal eosinophilia in subjects who are often middle-aged and who have perennial symptoms of sneezing paroxysms, nasal itching, rhinorrhoea and occasionally loss of sense of smell. However, they lack evidence of allergic disease as determined by skin tests and IgE levels. Some may represent an early stage of aspirin idiosyncrasy (Moneret-Vautrin et al., 1990). They often respond particularly well to treatment with intranasal corticosteroids (Jacobs et al., 1981; Mullarkey et al., 1979).

Nasal polyps may occur in association with cystic fibrosis (25% in children (Stern et al., 1982); 45% in adults (DiSant'Agnese & David, 1979)), asthma (30% (Moloney, 1977; Drake-Lee, 1984)) and as part of aspirin idiosyncrasy (Harnett, 1978; Spector et al., 1979) (acetylsalicylic acid sensitivity, sinusitis and asthma) but they most commonly occur alone. Infection, inflammation and imbalance of the arachidonic acid pathway or other metabolic pathways (Spector & Farr, 1983) have all been suggested as possible actiological factors. Allergy does not appear to predispose to polyp formation but mast cell reactions and eosinophil activation with subsequent inflammation seem to be important and may explain why corticosteroids are therapeutically effective.

Occupational rhinitis refers to rhinitis arising in response to an airborne agent present in the workplace. Causes include laboratory animals (rats, mice, guinea pigs, etc), grain (bakers and agricultural workers), wood dusts, particularly hard woods (mahogany, western red cedar, oreoko, etc), latex and chemicals such as acid anhydrides, platinum salts, glues and solvents (Schiffman & Nagle, 1992; Sataloff, 1992).

Hormonal rhinitis can occur during pregnancy (Mabry, 1986) and puberty and also in hypothyroidism (Incaudo & Schatz, 1991; Gupta et al., 1977) and acromegaly. Hormonal imbalance may also be respon-

sible for the atrophic nasal changes in post-menopausal women.

Drug-induced rhinitis can be produced by a number of medications (Meltzer et al., 1988). Reserpine, guanethidine, phentolomine, methyldopa, ACE inhibitors, and α-adrenoceptor antagonists such as prazosin have all been associated with nasal symptoms, as have topical ophthalmic β-blockers, chlorpromazine, aspirin, other non-steroidal anti-inflammatory agents and oral contraceptives (Ammat-Kohja, 1971). Classically, **rhinitis medicamentosa** results from long-term abuse with nasal decongestants, and drugs such as cocaine taken intranasally can produce significant irritation (Dax, 1990).

Food can produce rhinitis in a number of ways (Bock et al., 1978; Metcalfe, 1983). Gustatory rhinorrhoea (Raphael et al., 1989) may occur when eating hot and spicy foods, whilst specific allergic and hypersensitivity reactions may result to foods themselves or to colourants and preservatives. Alcohol produces a physiological vasodilatation and nasal congestion but can also provoke symptoms due to allergy and hypersensitivity to the many components contained in alcoholic drinks (Metcalfe, 1983). True food allergy almost never provokes isolated rhinitis symptoms and other organs are invariably involved in these responses.

Emotional factors such as stress and sexual arousal are known to have an effect on the nose, probably due to autonomic stimulation.

Primary atrophic rhinitis is a condition in which patients report nasal congestion, hyposmia and a constant bad smell (ozaena) in the nose (Zohar et al., 1990). It may be associated with headaches and chronic sinusitis. This persistent condition is characterized by progressive atrophy of the nasal mucosa and underlying bone of the turbinates (Goodman & DeSouza, 1987). The main finding is copious foul-smelling crusts filling the nasal cavity, which is usually capacious. It has been attributed to infection with a variety of bacteria including *Klebsiella ozaenae* (Henriksen & Gundersen, 1959) but these may be secondary contaminants rather than the primary pathogens. The syndrome should be distinguished from secondary atrophic rhinitis, resulting from chronic granulomatous infections, sinusitis, irradiation, radical nasal surgery and trauma.

Diagnostic techniques

The tests and procedures listed below represent the spectrum of investigations, only a small number of which are routinely available or indeed applicable to each individual patient.

History

General ENT examination

Allergy tests – skin tests
– total serum IgE
– serum specific IgE

Endoscopy – rigid
– flexible

Nasal smear – cytology

Nasal swab – bacteriology

Radiology – plain sinus radiograph
– CT
– MRI
– CXR

Mucociliary function – nasal mucociliary clearance (NMCC)
– ciliary beat frequency (CBF)
– electron microscopy

Nasal airway assessment – nasal inspiratory peak flow (NIPF)
– rhinomanometry (anterior and posterior)
– acoustic rhinometry

Olfaction – threshold testing
– 'scratch and sniff' tests

Blood tests – full blood count and white cell differential
– erythrocyte sedimentation rate
– thyroid function tests
– anti-neutrophil cytoplasmic antibody (ANCA)
– immunoglobulins and IgG subclasses
– antibody response to immunisation with protein and carbohydrate antigens

History and examination

A careful history will usually suggest the diagnosis of rhinitis. A thorough general medical history should be followed by questions specific to rhinological symptoms, including information on environmental and occupational factors and family history. Allergic rhinitis can occur at any age including infancy and the physician should note the onset of symptoms. Most patients with allergic rhinitis develop their symptoms prior to the age of 20 with a 15% incidence in young adults (Haahtela et al., 1980; Hagy & Settipane, 1969). The frequency of symptoms should be noted and whether they are daily, episodic, seasonal or perennial. The duration and severity of the symptoms should also be mentioned, and whether the severity has increased, decreased or remained the same over a period of time.

Patients with rhinitis can be divided into **'sneezers and runners'** and **'blockers'** (Mygind et al., 1982). Those with allergic rhinitis are more commonly **'sneezers and runners'**.

'sneezers and runners'
- sneezing, especially paroxysmal
- watery rhinorrhoea (anterior more than posterior)
- itchy nose
- nasal blockage (variable)
- diurnal rhythm (worse during day and improving during night)
- often associated conjunctivitis

'blockers'
- little or no sneezing
- thick nasal mucus (catarrh) more often posterior (postnasal drip)
- no itch
- nasal blockage often severe
- constant day and night but may be worse at night

Consistent obstruction on the same side suggests a polyp, structural problem or, rarely, a tumour. Hyposmia and anosmia are most often associated with nasal polyps or severe disease. Symptoms related to blockage of the airways include: frequent sore throats, dryness of the mouth and oropharynx, a nasal quality to the voice and snoring. An allergic salute may be characterized by an upward or sideways thrust of the palm of the hand against the tip of the nose when watery rhinorrhoea and itching are significant, resulting in a transverse crease in the supra-tip region of the external nose. If sneezing is present, it often occurs in paroxysms.

The allergens, irritants and weather conditions that precipitate or aggravate symptoms should be detailed. Perennial symptoms more commonly occur when there are mammals, dust mites or mould spores present

throughout the year. Moisture favours the growth of mites and moulds. Mattresses, pillows, curtains and carpets are frequent sources of dust mites. House plants and stored paper goods favour mould growth. There is a direct relationship between the amount of pollen exposure and severity of symptoms (Norman, 1985). As the season progresses, there is a gradual increase in severity of symptoms in relation to the pollen count due to immunologic enhancement of sensitivity (Levy & Osler, 1967) or 'priming' (Connell, 1969). Certain foods can induce rhinitis symptoms as has been confirmed by double blind challenges (Bock, 1987). Irritants can potentiate the symptoms of allergic rhinitis. Emotional upsets can also exacerbate rhinitis symptoms. In an allergic individual an upper respiratory tract infection can either mimic allergies or worsen or prolong the effects of allergies. Hormonal factors or medications such as topical vasoconstrictors, antihypertensives or cocaine can be responsible for a persistent rhinitis or even a rebound effect. A positive family history makes it more likely that an allergy will develop (Van Arsdel & Motulsky, 1959) but the pattern of inheritance seems to be polygenic and a negative family history by no means rules out the diagnosis of allergic rhinitis.

Examination of the nose is advisable in all cases of persistent or atypical/unilateral rhinitis. Ideally, this is accomplished with a nasal speculum and head light, otoscope with nasal adaptor, rigid Hopkins rod or fibre-optic nasopharyngoscope (Rohr et al., 1983).

The quantity and quality of the secretions should be noted, generally either clear and watery, or thick and discoloured. With abnormal mucociliary clearance or total nasal obstruction, thick secretions can be seen pooling in the floor of the nose. The mucosa is usually reddened in acute infections and over-use of topical medications, while the typical allergic mucosa appears pale and swollen though these distinctions are not absolute. A careful examination of the nasal cavity should enable the identification of polyps, tumours, foreign bodies or septal deflections. Unlike the nasal turbinates with which they are often confused, polyps appear glistening and opaque and are insensitive to touch. Crusting on an inflamed mucosa may suggest atrophic rhinitis or a systemic disease such as sarcoidosis. The presence of a septal perforation should raise the possibility of cocaine abuse, previous surgery or again systemic granulomatous diseases.

Physical examination should not be confined to the nose alone and should be accompanied by a full ENT examination, posterior rhinoscopy, indirect laryngoscopy, and palpation of the neck in selected cases. There may be fluid in the middle ear or other evidence of Eustachian tube dysfunction. With prolonged nasal obstruction and constant mouth breathing in childhood, an individual may have elevation of the upper lip, an overbite and a high arched palate (Bresolin et al., 1984). The eyes may be puffy, with cyanosis of the periorbital region (allergic shiners (Marks, 1963)), conjunctival injection, mucous discharge and increased tearing.

Examination of the lungs may reveal wheezing or a persistent cough since there are often accompanying symptoms of asthma when allergic rhinitis is present.

Table 1. Use of skin prick tests

- Diagnosis of atopy – the underlying predisposition to allergic disease
- Supportive evidence (positive or negative) for the clinical history
- Essential when expensive or time-consuming avoidance measures (house dust mite), or removal of a family pet, or consideration for immunotherapy are involved
- Educational value, providing a clear illustration to the patient which may reinforce verbal advice
- Practice points: always check that patient is not on antihistamines before performing skin tests. Always include positive (histamine) and negative controls
- Allergen-specific IgE (RAST) is an alternative if skin prick tests cannot be performed

Skin tests

Immediate hypersensitivity skin tests are used to demonstrate an IgE-mediated allergic reaction of the skin and represent the primary diagnostic tool in allergy. The use of skin prick tests is summarised in Table 1. If properly performed they yield useful confirmatory evidence for a diagnosis of specific allergy but there are many drawbacks in their performance. The potential for anaphylaxis dictates that they should only be performed under medical supervision by specially trained personnel when adrenaline (epinephrine) is available.

Methods. The prick and puncture test is recommended for the diagnosis of immediate type allergy in preference to scratch tests or intradermal skin tests. Although intradermal tests are more sensitive than prick tests, they may induce false positive reactions as well as some systemic reactions. Therefore they should only be used after skin prick tests are found to be negative. A wide variety of modified skin prick tests (SPT) are now available, the choice depending upon the skill, experience and preference of the investigator.

Cutaneous reactivity is highly variable and every skin test should include a negative and positive control solution. The negative control is the diluent used to preserve the allergen extracts which can provoke a reaction in patients with dermographism. Positive control solutions are either histamine or mast cell secretagogues such as codeine phosphate.

The immediate skin prick test induces a wheal and flare response that peaks in 10 minutes for histamine, 8–12 minutes for codeine phosphate and 15–20 minutes for allergens. Late phase responses may be observed but their significance is not fully understood. When there is no reaction to the negative control, small wheals with a mean diameter of over 3 mm with associated flare and itching represent a positive immunologic response, though they are not necessarily of clinical relevance. A large number of factors can affect the skin prick reaction (Table 2).

Carefully performed and correctly interpreted skin

Table 2. Factors affecting skin testing

Drug	
Antihistamines	
First generation (sedating)	(2–4 days)
Second generation (non-sedating	
astemizole	(6–8 weeks)
others	(1 week)
Ketotifen	(1 week)
Imipramine	(4 weeks)
Phenothiazines	(48 hours)
Corticosteroids	
locally on test site (2–3 months)	
Specific immunotherapy	
Site of skin test	(flexor aspect of lower arm, avoiding wrist)
Age	(response smaller in very young and old)
Seasonal variations	(response larger after season)
Pathologic conditions	(response increased in acute urticaria, dermographism; decreased in atopic dermatitis and chronic haemodialysis)

tests with high quality allergen extracts covering relevant allergens in the patient's environment are a simple, painless and inexpensive method with a high efficiency/cost ratio. Performance of a large number of allergen skin prick tests is expensive, time-consuming and usually unnecessary, particularly in the primary care setting. In general, the number of skin tests that are routinely performed need only be limited to common aeroallergens in the patient's environment (e.g., approximately six; to house dust mite, pollens, moulds and domestic pets). The skin prick test represents the primary tool for allergy diagnosis by a trained physician. However, the occurrence of positive responses does not necessarily imply that the patient's symptoms are due to an IgE mediated allergy since skin tests are positive in 10–15% of symptom-free individuals. Skin prick testing for inhaled allergens is of greater clinical reliability than in the case of foods and industrial sensitization.

IgE

The discovery of IgE in 1967 was a major advance in the understanding and diagnosis of allergic diseases. However, at present, *in vitro* methods based on IgE determination are not superior to skin tests in most cases.

Total serum IgE is measured by a variety of radio- or enzyme-immunoassays. In normal subjects, levels of IgE increase from birth (0–1 kU/l) to adolescence and then decrease slowly to reach a plateau after the age of 20–30 years. Levels over 100–150 kU/l are considered raised and result from a number of conditions as well as allergy. In addition over 50% of seasonal allergic rhinitic patients have normal total IgE; thus the titration of total serum IgE is only poorly specific and of limited use.

Serum specific IgE, in contrast to total serum IgE, may be helpful particularly when extracts for skin prick tests are not available. A variety of methods is available, of which the radioallergosorbent test (RAST) and those using radio- or enzyme-labelled anti-IgE are widely used. Results are expressed as total radioactive counts bound (cpm), arbitrary units (RAST class, PRU/ml) or units of IgE (IU/ml). In addition to these tests based on single allergens bound on a solid phase, others exist using double-antibody assays with liquid systems. Using standardised allergen extracts, measurement of serum specific IgE correlates well with skin prick tests and nasal challenge but false positives and negatives may occur. It is, however, unusual to have a low specific IgE in the presence of allergic symptoms.

A number of screening tests using several allergens on a single solid phase or testing several allergens during a single assay are also available. Their efficiency (both in specificity and sensitivity) in allergy diagnosis can be over 85%, defining those individuals who require more detailed investigation but it must be remembered that important regional allergens may not be considered in such screening tests.

Nasal smears – cytology and histology

Nasal smears may differentiate between allergic and infectious rhinitis. Eosinophils are characteristic of allergy and non-allergic rhinitis with eosinophilia syndrome (NARES) whereas neutrophils imply bacterial infection.

Other tests

Release of mediators from peripheral blood cells

Blood basophils of allergic patients can degranulate and release mediators (histamine and leukotrienes) when stimulated by a specific allergen. The assay of histamine or leukotriene release or the microscopic examination of cells (e.g., basophil degranulation test) can be performed but they are research tools only.

Mediators released during allergic reactions

The measurement of mediators and enzymes released in peripheral blood, nasal secretions or urine during an allergic reaction has been made possible by the development of highly specific and sensitive immunoassays for the titration of histamine, PGD_2, $LTC_4/D_4/E_4$, tryptases, kinins and eosinophil cationic protein. These can be measured at resting baseline levels or after allergen challenge but at present remain research tools.

Nasal challenge

Nasal challenges with allergen are of value in research but are time-consuming and potentially dangerous especially if the patient is asthmatic. Histamine or methacholine may also be used to estimate the level of non-specific reactivity.

Interpretation of allergy tests

The diagnosis of allergy is based primarily on a thorough clinical history which may correlate with other investigations but cannot be based alone upon the results of skin prick tests, *in vitro* tests or nasal challenge. Skin prick tests represent the primary diagnostic tool used for immediate-type hypersensitivity. Comparison between the titration of specific IgE and skin tests depends upon the quality and standardisation of allergens used in both tests and, to a lesser extent, on the method of skin testing used. The worst correlations have been obtained with house dust,

mould, food extracts and unstandardised dander extracts. There are good correlations between a strongly positive response to a skin test and the detection of serum specific IgE and between a negative response to a prick test and the lack of detection of serum specific IgE, whereas small wheals induced by prick tests and positive results of intradermal tests only at high concentrations of extracts are less frequently associated with the detection of serum specific IgE. Positive response to skin tests and serum specific IgE can be found in totally symptom-free subjects with a similar prevalence.

Correlations between responses to skin prick tests and serum specific IgE with inhalation challenges are less consistent because of the nonspecific hyperreactivity.

Endoscopy

ENT examination in the clinic is now considerably facilitated by the use of rigid Hopkins rods and flexible fibre-optic endoscopes. Administration of topical intranasal anaesthesia is recommended at initial assessment. Specific attention is paid to abnormality within the middle meatus and nasopharynx.

Bacteriology

Specimens obtained under direct endoscopic visualisation from the middle meatus correlate well with formal sinus aspirates, in contrast to routine swabs taken blindly from the nose and nostril.

Imaging

Plain sinus radiographs. Gross opacification, mucosal thickening and bone erosion of the maxillary, frontal and sphenoid sinuses may be demonstrated. The lateral nasal wall and the ethmoid labyrinths are poorly visualised by conventional radiographs and false negatives and positives may frequently occur.

Computerised tomography (CT). This has become the principal radiological investigation for major sinonasal disorder but is of limited use in the diagnosis of allergy, except to eliminate other conditions (Lloyd et al., 1991). By demonstrating soft tissue and bone detail, it defines the extent of disease, often indicating the diagnosis when certain characteristic features are present and offers the optimum demonstration of surgical anatomy. Various protocols are available, utilising wider and narrower window widths to image bone and soft tissue respectively. Contiguous sections at 0.5 mm intervals or less are obtained directly in coronal and axial planes. Reformatting should be reserved for sagittal views only and when patients cannot sufficiently extend the head. The use of a topical vasoconstrictor before scanning can be helpful in distinguishing physiological mucosal swelling from pathologic change (Stringer et al., 1993).

Magnetic resonance imaging (MRI). This technique relies upon the principle that hydrogen nuclei align their magnetic axes parallel to a static magnetic field. The addition of paramagnetic contrast agents such as gadolinium-DTPA increases the ability to differentiate normal from pathological tissue and secretion. This combined with imaging in three planes makes it the modality of choice in neoplasia. However, the absence of bone detail (present as a signal void) limits its usefulness in inflammatory and infectious sinonasal conditions.

Chest radiograph. A chest radiograph may be indicated when lower respiratory symptoms are present suggestive of primary mucociliary problems or systemic disease such as sarcoidosis or Wegener's granulomatosis.

Mucociliary function

Nasal mucociliary clearance. A simple test of the system can be performed by placing a 0.5 mm piece of saccharin on the anterior end of the inferior turbinate 1 cm from the end and the time taken to taste something sweet in the mouth measured (Andersen et al., 1974). Normally this occurs within 30 minutes. If longer than an hour has elapsed, it is worth repeating the test in case the particle has fallen out and checking that the patient is capable of tasting saccharin.

Ciliary beat frequency. When the saccharin test is prolonged or if specific ciliary abnormalities are suspected it is possible to examine the cilia directly by taking a sample with a bronchoscopy cytology brush and observing cilial activity under a phase-contrast microscope with photometric cell (Rutland et al., 1982). The frequency can be measured with a real-time analyser and expressed in hertz, the normal range from the inferior turbinate being 12–15 Hz. This technique is presently only available in a few centres.

Electron microscopy. If the nasomucociliary clearance time and ciliary beat frequency are abnormal, samples may be obtained by cytology brushing or biopsy for electron microscopy studies to diagnose conditions such as primary ciliary dyskinesia.

Nasal airway assessment

Nasal expiratory or inspiratory peak flow (NIPF). This technique, which uses a peak flow meter, has the advantage of being inexpensive, quick and easy to perform; it is useful for repeated examinations and

compares well with rhinomanometry (Holmstrom et al., 1990). Of the two methods, forced inspiration is preferred although it can produce significant vestibular collapse. Forced expiration inflates the Eustachian tube, which the patient may find distressing, and can produce an unpleasant quantity of mucus in the mask.

Rhinomanometry. Rhinomanometry attempts to measure nasal airway resistance by making quantitative measurement of nasal flow and pressure (Clement, 1984). It employs the principle that air will only flow through a tube when there is a pressure differential, passing from areas of high to low pressure, a differential created by respiratory effort. When the nasal mucosa is decongested, the reproducibility of rhinomanometric results is good but it is time-consuming and is mostly a research tool. Active anterior rhinomanometry is most commonly used, as the posterior technique cannot be used in 20–25% of individuals due to an inability to relax the soft palate.

Acoustic rhinometry. In this technique an audible sound pulse (150–10000 Hz) generated by a spark is propagated in a sound tube and is passed into the nose where it is altered by variations in a cross-sectional area. The reflected signal is picked up by a microphone and analysed. It is thus possible to determine the area within the nasal cavity as a function of distance and from this volumes may be derived (Hilberg et al., 1989). It does not measure airflow. The technique is still being evaluated but it allows accurate sequential quantification of abnormalities, pre- and post-treatment.

Olfaction

Olfactory thresholds. Estimation of olfactory thresholds may be established by presentation of serial dilutions of pure odorant such as pm-carbinol (Amoore, 1992). The patient is presented with two bottles, one containing only the diluent solution as the control, the other the odorant in progressively increasing or decreasing concentrations, and each is sniffed in turn. The point is reached at which the patient cannot distinguish between the control and test bottle, and this indicates the minimum detectable odour.

Other quantitative olfactory testing. 'Scratch and sniff' tests using patches impregnated with microencapsulated odorants are available (Doty et al., 1984). The patient is forced to choose between a number of options after scratching the patch to release the odour and the results take into account answers guessed correctly and deliberately given incorrectly.

Blood tests

When considering the wide variety of local and systemic conditions which may present as 'rhinitis', it may be necessary to perform a number of haematological tests to elucidate the differential diagnosis, e.g., thyroid dysfunction, Wegener's granulomatosis and immune deficiency.

Treatment of allergic rhinitis

Allergen avoidance

Environmental control measures (Colloff et al., 1992)

In industrialised countries, most of the time is spent indoors and this environment contains important triggers of rhinitis. The outdoor and indoor allergic and non-allergic environment has changed within the past 30 years in most countries and has probably increased the risks of allergic diseases. In temperate climate zones, energy saving has led to air-tight houses with low natural ventilation and has increased the number of mites.

When possible, environmental control measures for indoor allergens should be applied even if their efficacy is not complete, as they may generally improve the patient and reduce the need for pharmacologic treatment. Furthermore, even when an allergen source such as a pet is removed from a patient's environment, the benefit may take several weeks or months to be perceived. Mite avoidance can be attempted by reducing the amount of house dust itself, by reducing indoor humidity, by the application of acaricides and by the use of mite-proof covers. It is more difficult to reduce exposure to pollens and outdoor allergens.

POLLEN AVOIDANCE MEASURES

- monitor pollen forecasts
- avoid high pollen areas
- stay inside house when pollen count is high
- keep windows and doors closed when pollen count is high
- use high efficiency particulate (HEPA) filters in cars
- consider using glasses outside house

HOUSE DUST MITE CONTROL MEASURES

The bedroom
- use allergen-impermeable mattress, duvet and pillow covers on all beds in room
- thoroughly vacuum the mattress, pillows, around the base of the bed and bedroom floor each week
- remove feather pillows, woollen blankets and eiderdowns – replace with synthetic ones and wash them weekly at 60°C
- remove carpeting if possible
- wipe all surfaces each week including pelmet tops, window sills and tops of cupboards with a damp cloth
- have light washable cotton curtains and wash frequently
- use a vacuum cleaner with disposable paper bags and a filter or a vacuum with a water-reservoir. Wear a mask whilst cleaning or preferably get someone else to do it
- chemical agents (acaricides) to reduce house dust mite population may be helpful

Other rooms
- particular attention should be directed at removal of dust from upholstered furniture. Vacuum clean at least twice a week, including headrests, arms and edges of seats

Children
- affected children should be out of the room when cleaning is being done and should not return for two hours
- children should not sleep with furry toys in their beds. Toys should be vacuumed, tumble-dried or put in the deep-freeze (−20°C) overnight to reduce mites

Pets
- remove pets (if possible)
- do not replace animal
- no pets in the bedroom at any time. Allergic families should avoid having furred or feathered pets since allergic sensitivity to them may develop in time, even if not immediately apparent
- wash pet regularly

Pharmacological principles

In selecting medication, the aim of that therapy should be considered in relation to the aetiology and pathophysiology of the condition. If it is possible to anticipate the likely onset of symptoms, as in seasonal rhinitis, it is advantageous to commence medication prophylactically, thus suppressing symptoms from the outset rather than waiting until they are apparent.

receptors and had varying degrees of anticholinergic activity. Some compounds had additional antiserotonin, antibradykinin activity (Meltzer, 1990). Antihistamines primarily inhibit symptoms caused by endogenous histamine release and do not seem to influence the priming effect of allergenic exposure. They seem to block the development of hyper-responsiveness but not the cellular influx of eosinophils into the mucosa during the pollen season (Klementsson et al., 1989).

Treatment of allergic rhinitis in adults *

	Itch/Sneezing	Discharge	Blockage	Impaired smell
Sodium cromoglycate	+	+	±	–
Oral antihistamines	+++	++	±	–
Ipratropium bromide	–	+++	–	–
Topical decongestants **	–	–	+++	–
Topical corticosteroids	+++	+++	++	+
Oral corticosteroids	+++	+++	+++	++

* Match drug profile to patients' symptoms and always include allergen avoidance advice where appropriate.

** Restrict use of topical decongestants to 7 days. In USA, (oral) decongestants may be an alternative.

Antihistamines

Histamine is an important mediator released from mast cells and basophils. Three different histamine receptors have been described (H_1, H_2 and H_3) (Ishikawa & Sperelakis, 1987). Histamine stimulation of blood vessels results in dilatation and increased permeability (H_1) (White et al., 1987). Stimulation of sensory nerve endings in the nose results in itching, sneezing and secretion (H_1) (White et al., 1987). Antihistamines bind to the histamine receptor without giving rise to stimulation (competitive antagonism). Thus antihistamines are effective in reducing established symptoms such as nasal itching, sneezing and watery rhinorrhoea and may be more effective when taken prophylactically but have little objective effect on nasal blockage (Brooks et al., 1990).

The varying sedation associated with the early antihistamines limited their use. This effect is CNS dependant and partly related to their anti-emetic effect and their ability to reduce motion sickness. The majority of antihistamines had affinity for muscarinic

The newer less-sedative or non-sedative antihistamines differ in their pharmacokinetic properties. Astemizole has a markedly long half-life (10 days in serum), reducing skin prick reactions to histamine and allergens for 6–8 weeks (Felderman & Rosen, 1987). It can give rise to weight gain in some individuals.

Cetirizine, loratadine, acrivastine and terfenadine are metabolised more rapidly, so the suppressive effect on skin reactions is present for 2–4 days after drug ingestion. Cetirizine differs from other antihistamines in that it is not metabolised in the liver and no active metabolites are generated, so it is mainly excreted in the urine (Campoli-Richards et al., 1990).

Antihistamines have usually been taken orally, either as tablets or syrup, which has the advantage of reducing systemic symptoms such as conjunctivitis and urticaria. Comparative studies between oral antihistamines are difficult to evaluate. Full dose-response curves have not been performed in the same individuals to compare efficacy and side-effects. Although the newer antihistamines are more expensive than the earlier preparations, the cost is substantially offset by

their diminished sedative effect and lack of effect on performance.

Serious cardiac side-effects have been observed with some antihistamines. All antihistamines should be given in the recommended doses, and terfenadine and astemizole should not be given in conjunction with macrolide antibiotics (e.g., erythromycin) and some oral antifungal agents (e.g., ketoconazole and itraconazole) or in the presence of significant liver disease. At present it is not known whether this side-effect is due to a class-effect of antihistamines.

Terfenadine is effective and safe, except in the following circumstances where a risk of ECG QT prolongation and potential for ventricular arrhythmia exist:

- **do not exceed recommended dose (120 mg daily in adults)**
- **avoid drug interactions (ketoconazole and erythromycin)**
- **do not use in presence of significant liver disease.**

Topical antihistamines such as azelastine and levocabastine are now available and can be used for acute symptomatic relief and prophylaxis of allergic rhinitis, without any systemic side effects. (Bende & Pipkorn, 1987; Holmberg et al., 1989, Davies et al., 1993).

Although H_2-receptors exist in nasal blood vessels, their importance in rhinitis is of limited importance and the use of H_2-antagonists cannot be generally recommended (Secher et al., 1982).

Corticosteroids

The mechanism of action of corticosteroids results from the steroid molecule which penetrates the cell membrane and binds to hormone receptors in the cytoplasm. The steroid receptor complex is transferred to the nucleus where binding occurs to specific sites on the DNA molecule. These sites have a regulatory influence on protein synthesis which may be increased or decreased by this binding (Pauwels, 1986). Of clinical importance in rhinitis, corticosteroids reduce inflammatory cell infiltration, in particular decreasing the number of mast cells (Pipkorn, 1983; Gomez et al., 1988) and eosinophils in the superficial layers of the nasal mucosa, diminish hyperreactivity and vascular permeability and may reduce the release of mediators from mast cells (Pipkorn et al., 1987; Svensson et al., 1990).

Corticosteroids may be delivered topically to the nose, taken orally or given parenterally. Modern topical steroids have a considerably increased therapeutic ratio. This results in an enhanced beneficial local effect whilst systemic effects are minimised. Beclomethasone dipropionate (BDP) was introduced in 1973, combining a high topical efficacy with rapid deactivation in the liver (Mygind, 1973). Subsequently, flunisolide, budesonide, triamcinolone and fluticasone propionate have become available. In individual cases, higher doses may be beneficial for a short period to control symptoms. These drugs can be a useful alternative to systemic corticosteroids. Once symptoms are controlled, the daily dosage may be reduced. Recommended frequency for BDP, flunisolide and budesonide is twice daily; for fluticasone propionate and triamcinalone, it is once a day.

Topical corticosteroids are administered by Freon-driven aerosols and by mechanical pump sprays in aqueous or glycol solutions, or as a dry powder. The patients should be carefully instructed on the method of usage to obtain optimum distribution. Occasionally, crusting, dryness and slight spotting of blood may occur and glycol may cause slight discomfort in some individuals. These minor problems may be overcome by changing the delivery system; for example, to an aqueous form (Mygind, 1993). Exceptionally, septal perforation has been reported in association with intranasal steroid preparations (Soderberg-Warner, 1984).

Topical corticosteroids are effective in reducing nasal blockage, itching, sneezing and rhinorrhoea in allergic and non-allergic non-infectious rhinitis (Orgel et al., 1991). Their ability to reduce nasal blockage and efficacy in non-allergic rhinitis gives them an advantage over systemic antihistamines. They have proved more effective in symptomatic control of allergic rhinitis than sodium cromoglycate, (Welsh et al., 1987), antihistamines (Harding & Heath, 1976) and decongestants (Juniper et al., 1989). However, initially in very congested noses, a topical decongestant, such as oxymetazoline, is often advantageous before instilling the intranasal steroid.

The topical intranasal preparations, betamethasone sodium phosphate drops and dexamethasone spray are also very effective in symptomatic relief of allergic and non-allergic rhinitis. They are, however, capable of producing minor systemic steroid effects so their long-term use is not recommended.

Short courses of systemic corticosteroids can be of use in urgent or severe cases of rhinitis, but should only be used with caution and when there are no contraindications. These would include herpes keratitis, advanced osteoporosis, severe hypertension, diabetes mellitus, gastric ulceration and chronic infection. Systemic steroids should not be used for rhinitis in children or during pregnancy. Whenever possible, other anti-rhinitic medication should be given concomitantly to diminish the dosage of the steroid and to continue symptomatic control after the systemic steroids have ceased their action.

Systemic steroids may be given orally (e.g., prednisolone, dexamethasone) or as a depot injection of a micro-crystalline ester (e.g. methylprednisolone). The depot preparations, although effective, should be dis-

couraged as they can produce severe side-effects, cannot be reversed and suppress adrenal cortex function for long periods (Hedner & Persson, 1970).

Sodium cromoglycate

Sodium cromoglycate (cromolyn sodium) is used topically and was thought to inhibit IgE-dependent allergic reactions by reducing the release of histamine and other mediators from mast cells (Okuda et al., 1985). Sodium cromoglycate binds to receptor-like protein in the cell membrane, called cromoglycate binding protein (Dixon et al., 1980). This site is closely associated with mechanisms that control calcium movement in membrane. Sodium cromoglycate binding to a cell is accompanied by phosphorylation of the 78-kDa protein which occurs simultaneously with the inhibition of IgE-dependent degranulation. Sodium cromoglycate reduces nasal itching, sneezing, hypersecretion and nasal blockage in allergic rhinitis. It has negligible side-effects but its duration of therapeutic efficacy means that it should be applied at least four times daily; this can lead to poor patient compliance. Sodium cromoglycate is primarily a prophylactic drug for use in children, but it is less effective than topical corticosteroids.

Anticholinergics

The anticholinergics, ipratropium bromide and oxitropium bromide inhibit muscarinic cholinoceptors. Cholinergic receptors are of importance in the production of nasal secretion but have little or no role in vascular control (Raphael et al., 1991). Thus anticholinergics can reduce watery rhinorrhoea, which may occur in a number of forms of rhinitis, but they have no effect on nasal blockage (Mygind & Borum, 1990). Nasal application of ipratropium bromide markedly inhibits methacholine induced and cold-air induced nasal hypersecretion (Borum, 1978). They have no influence on sensory nerve endings and thus no effect on nasal itching or sneezing (Jokinen & Sipila, 1983).

Decongestants (α-adrenoceptor agonists)

α_1-adrenergic receptors are located on the postsynaptically located effector organ; e.g., on vascular smooth muscle. These receptors are naturally stimulated by noradrenaline and adrenaline. α_2-receptors are located on both presynaptic and postsynaptic nerve endings. Stimulation of presynaptic receptors reduces release of noradrenaline (Lacroix, 1989). α-adrenergic vasoconstrictors reduce nasal obstruction but have no influence on itching, sneezing or nasal secretion.

The most commonly used topical α_2-adrenergic receptor stimulators are imidazoline derivatives such as oxymetazoline, xylometazoline and naphazoline. The nasal administration of local vasoconstrictors is not usually recommended for more than 7–10 days because of the risk of inducing rhinitis medicamentosa (Proctor & Adams, 1976). This condition may occur because of down-regulation of α-adrenoceptors which makes them less sensitive to endogenously released noradrenaline and exogenously applied vasoconstrictors.

Ephedrine, pseudoephedrine and phenylpropanolamine are given orally and have both α_1- and α_2-adrenoceptor agonistic activity; furthermore, ephedrine has an effect on β-adrenoceptors. Phenylephrine is used topically and has α_1-adrenergic receptor activity.

In a dose-ranging study of phenylpropanolamine it was shown that 25 mg was significantly more effective than placebo or a 15 mg dosage in objectively improving nasal airflow (Darnansjah et al., 1990). This effect was maximal at one to one and a half hours and maintained for two hours. A combination of phenylephrine, phenylpropanolamine and guaiphenesin has been shown to significantly reduce nasal symptoms, compared to placebo, with minimal side-effects (Erffmeyer et al., 1982).

The oral formulations such as pseudoephedrine in sustained release preparations have gained some popularity combined with antihistamines such as terfenadine (Howarth et al., 1993; Henauer et al., 1991). This combination relieves both the nasal obstruction and itching, sneezing and rhinorrhoea (Storms et al., 1989). In a study with acrivastine, pseudoephedrine and the combination in seasonal allergic rhinitis, the combination was significantly better than either placebo or pseudoephedrine alone in controlling symptom scores, and it was superior to acrivastine alone in controlling all symptoms except itchy eyes (Meran et al., 1990).

The systemic use of adrenoceptor agonists may result in side-effects such as restlessness, agitation, sleep disturbances, tachycardia, angina pectoris, hypertension, headache and micturition problems (Änggård & Malm, 1984). Oral adrenoceptor agonists should not, therefore, be used in cases of coronary heart disease and should be avoided in cases of thyrotoxicosis, glaucoma and diabetes.

Saline douching

Clinical studies have shown the beneficial effects of regular nasal douching with saline (Spector et al., 1982).

Immunotherapy

The efficacy of specific immunotherapy (SIT) for pollen (grass (Varney et al., 1991), ragweed, birch, mugwort, olive, mountain cedar pollen and *Parietaria* (Or-

IMMUNOTHERAPY WITH OTHER AGENTS

With the general availability of dust mite extracts, house dust extract need no longer be used for immunotherapy

Specific immunotherapy with extracts of undefined allergens (bacteria, food, insects, etc) should not be used.

Practical use of immunotherapy

a) SIT should be considered if
- pharmacotherapy insufficiently controls symptoms or produces undesirable side-effects
- appropriate avoidance measures of indoor allergens fail to control symptoms
- there is a history of allergic rhinitis for at least two seasons (seasonal) or six months (perennial)
- there are positive skin tests or serum specific IgE which correlates with rhinitis symptoms

b) Contraindications to SIT include
- concomitant therapy with a beta-blocker
- contraindication to the administration of adrenaline (epinephrine)
- non-compliance by patient
- autoimmune disease
- induction but not maintenance therapy during pregnancy
- uncontrolled asthma (Malling & Weeke, 1993) (In the UK, immunotherapy is not currently recommended for any patient with asthma (BSACI Working Party Position Paper on Allergen Immunotherapy, 1993))

c) When SIT is indicated, a complete discussion with the patient to include the potential dangers as well as the optimal duration of therapy should be done prior to initiation of therapy

d) SIT should be prescribed by a specialist and administered under the supervision of a physician trained to manage anaphylaxis. Adrenaline (epinephrine), corticosteroids and equipment for respiratory resuscitation must be readily available (Malling & Weeke, 1993).

tolani et al., in press), house dust mite (Blainey et al., 1984; Corrado et al., 1989; Ewan et al., 1988), and cat dander allergy have been clearly documented in double blind controlled trials. Severe symptomatic reactions occasionally may occur, especially in asthmatic patients. In certain countries (the UK and the Scandinavian countries) the use of SIT has been greatly curtailed due to adverse reactions, but the injections were given by non-specialists. To minimise risk and improve efficacy, SIT must be prescribed by specialists and administered under the supervision of physicians trained to manage systemic reactions and with the immediate availability of adrenaline (epinephrine) should anaphylaxis occur. All patients should be observed for at least 30 minutes following allergen injections. Patient selection is important and efficacy must be balanced against potential side-effects.

The efficacy of SIT is dose-dependent. Low doses are usually ineffective and should not be used any longer. On the other hand, the 'highest tolerated dose' may increase the risk of severe systemic reactions and should be replaced by the 'optimal maintenance dose', i.e., a clinically effective dose giving a low and acceptable rate of mild systemic reactions.

Future therapies

As the mechanisms of rhinitis become better understood, new therapeutic strategies might include the use of specific anti-cytokine antibodies directed against important cytokines such as IL-4 and IL-5. Another possibility may be the use of topical interferon-gamma. The recent availability of bradykinin antagonists not only should clarify the role of bradykinin in allergic rhinitis but may have therapeutic potential. A further possibility is the development of more potent topical corticosteroids with an even lower potential for systemic side-effects. In selected cases desensitization with aspirin and other non-steroidal anti-inflammatory agents may be appropriate. Modifications of immunotherapy are currently being evaluated. For example, the development of non-stimulatory peptides may be effective in reducing the risks of anaphylaxis by altering T-cell responsiveness whilst avoiding mast cell activation.

Compliance and patient education

Treating rhinitis requires more of the clinician than simply prescribing the appropriate medication. Rhinitis is a condition in which patient understanding, participation and compliance will contribute greatly to overall treatment effectiveness. Therefore, an essential first step in the education of the patient must be the development of a trusting relationship between clinician and patient. Initially time should be taken to explain the basic pathology of rhinitis and why it develops. This leads naturally to guidance on avoiding or reducing exposure to relevant allergens. Time is well-spent explaining how specific medications work, thus enabling patients to adjust their therapy appropriately for themselves in the future. Patients need to be shown how to use nasal applicators and to be told what to do if side-effects occur. Simple written instructions for both prophylactic and active use of medications can help the patient to self-medicate early and appropriately in the future. In cases of apparent treatment failure, compliance and correct use of medications should always be checked. A recent study has shown that if patients are provided with appropriate medications, education and written instructions before a pollen season, symptoms can be well controlled with minimum impairment to quality of life (Juniper et al., 1992).

Special considerations

Rhinitis in children

The pathophysiologic mechanisms, the clinical picture and the diagnostic and therapeutic aspects of rhinitis in children are virtually the same as those in adults. There are, however, some differences that justify a special consideration (Mygind, 1991; Siegel, 1993; Van Cauwenberge, 1991).

Infectious rhinitis is usually one of the first diseases in life. Viral rhinitis can occur in the first few weeks but becomes more frequent with exposure to other children. The mean frequency of the common cold in children 2–6 years of age, is 6 per year. Secondary bacterial rhinitis may prolong infection from a few days to several weeks. The sinuses should be regarded as integral with the nasal cavity and thus inflammation produces rhinosinusitis. The ethmoidal sinuses are already well developed at birth, while the maxillary sinuses gradually increase in size from birth to adolescence but are well developed at three years of age. The sphenoid and in particular the frontal sinuses are well visualized from 10 to 14 years.

In various epidemiological studies it was shown that allergic diseases can be observed from birth until death. However, the type of allergic reaction, its expression and the localisation will differ according to age. In the first months of life, dermatological problems such as constitutional eczema are predominant and are usually an early sign of a predisposition to allergy. In addition, in the early years of life recurrent attacks of bronchitis or bronchial wheezing are frequently caused by an underlying allergic disorder.

During infancy and early childhood, foods are often the offending allergen, while inhaled allergens become more important with increasing age. Until the age of 4, a clinical manifestation of IgE-mediated allergy in the ears, nose and throat is rare. The obvious expression of allergic rhinitis is not seen until after 4 or 5 years. Thereafter there is a progressive increase in the incidence of allergic rhinitis to reach 10 to 15% in adolescents. The most typical manifestation of allergic rhinitis is seen in adolescents and young adults. In the young child, allergy may underlie recurrent infectious rhinosinusitis, adenoiditis, otitis media and tonsillitis but may be overlooked.

Non-allergic non-infectious nasal hyperreactivity and non-allergic rhinitis with eosinophilia syndrome (NARES) are infrequent in children and usually only occur in adulthood. Similarly children rarely suffer from nasal polyps. When they do occur, cystic fibrosis or primary ciliary dyskinesia must be considered in children, whereas aspirin intolerance (idiosyncrasy) occurs more commonly in adolescents and adults than in children.

Diagnosis

Sometimes a child with allergic rhinitis can be recognised by some facial characteristics and mannerisms, such as the 'allergic salute', the allergic crease, 'Dennie's line' and infraorbital dark circles or 'allergic shiners'. None, however, are pathognomonic. A general ENT examination, which in the older child may include flexible or rigid rhinoscopy, will allow exclusion of other pathologies such as infection, polyp or tumour rather than confirm allergy. The classical description of an allergic nasal mucosa as swollen and pale with watery rhinorrhoea is typically present only when the patient has current symptoms at the time of examination. Other clues may include serous otitis media and adenoidal hypertrophy.

Although no child is too young to undergo a skin test, they are less reliable in those under two years of age. The normal values of total and specific serum IgE are lower than in adults, becoming proportionally lower with decreasing age. It should also be remembered that total serum IgE is only elevated in less than half of the children with allergic rhinitis. Nasal challenge is rarely performed in children as it is unpleasant and does not provide any useful additional information.

A nasal smear may provide information about the involvement of polymorphonuclear cells, as in an infectious process, or eosinophils, though these may be found in both allergic and non-allergic nasal hyperresponsiveness. As in adults, peripheral blood eosinophilia is only occasionally elevated in children with allergic rhinitis.

Therapeutic considerations

The principles of treatment of rhinitis in children are the same as those in the adult, though the emphasis may differ somewhat.

Prophylactic measures of allergen avoidance assume an even greater importance in children with allergic rhinitis. It has been shown that where an hereditary predisposition to allergy exists, the clinical symptoms only manifest after significant exposure to the allergens. However, it is possible that early intervention with allergen avoidance or immunotherapy may modify the course of disease in susceptible children. Controlled studies are required.

Oral antihistamines, especially the newer non-sedating varieties, are primary agents in the treatment

of allergic rhinitis (Simons, 1993). In young children it may be more difficult to administer topical medications due to lack of co-operation. Once or twice daily dosage is preferable, allowing medication to be given at home rather than involving the school staff.

Sodium cromoglycate nasal spray has been used for many years and can be prescribed in very young children but its use does require co-operation. For optimal benefit sodium cromoglycate should be given at least four times a day, but such a regime is often associated with poor patient compliance.

Some physicians are reluctant to prescribe topical nasal steroids in children because of concerns about the long-term systemic and local effects of these drugs. However, after over 15 years' experience with topical nasal steroids, there are virtually no adverse reports and they may, therefore, be used at the prescribed dosage (generally half the adult dose preferably given once daily in the morning), particularly where nasal obstruction is the most pronounced symptom.

Systemic corticosteroids have virtually no place in uncomplicated allergic rhinitis in children. It is dangerous to give topical vasoconstrictors in children under one year of age, due to the narrow range between therapeutic and toxic dose. The latter can result in cardiovascular and central nervous system disturbances. Oral decongestants should also be prescribed with care as they occasionally produce central effects.

In summary, treatment of allergic rhinitis in a child should depend predominantly upon prevention and be therapeutically as unaggressive as possible.

Rhinitis in the elderly

Allergic mechanisms are rarely the cause of perennial rhinitis in people over 65 years of age. In this population, it is generally non-allergic mechanisms such as autonomic imbalance, an alteration in muscarinic receptors, or the sequelae of earlier nasal disorders and their treatment which are responsible.

One of the best examples of nasal hyperresponsiveness in the elderly is 'old man's drip', a clear profuse watery rhinorrhoea which forms a dewdrop at the end of the nose. Various mechanisms have been suggested. Ipratropium bromide may be of some benefit (Mygind & Borum, 1990).

Genuine allergic rhinitis may occur but age has some implications for therapy. H_1-antihistamines are safe in the elderly but the older antihistamines may cause bladder disturbances, such as retention and problems of visual accommodation. Vasoconstrictors, especially when taken orally, more frequently give rise to cardiovascular and CNS side-effects (Malm & Änggård, 1993).

Rhinitis in pregnancy

Rhinitis symptoms such as nasal obstruction and rhinorrhoea may occur during pregnancy, predominantly from the second month to term. They usually disappear rapidly after delivery. Both oestrogens and progesterone cause increased activity of the seromucous glands, and an increase in nasal blood volume results in nasal congestion. Sufferers of allergic rhinitis may find their symptoms altered, for either better or worse, during pregnancy (Schatz & Zeigler, 1988).

Treatment must obviously be given with care. Teratogenic effects of the older antihistamines were noted in animals but have not been reported with the newer more specific H_1-antagonists. However, some caution is still required as the availability of the new generation antihistamines is still relatively recent, and they are known to be excreted in breast milk.

Oral decongestants theoretically may cause vascular disturbances in the placenta and foetus, but, given within the recommended dosage, oral pseudoephedrine is approved for use in pregnancy and widely used in the United States (Middleton et al., 1988). Saline douche and sodium cromoglycate may be tried initially, and if they are unsuccessful, topical nasal corticosteroids given at the recommended dosage have not been associated with any teratogenic or other adverse effects.

Subcutaneous immunotherapy should not be commenced during pregnancy but maintenance therapy may continue if it is proving effective (Bousquet & Michel, 1993; Metzger et al., 1978).

Rhinitis in athletes

Physical exercise is a potent vasoconstrictor. The nasal resistance decreases gradually with increasing pulse, due mainly to the release of noradrenaline. In all cases of nasal obstruction related to vasodilatation, physical exercise will increase patency though the individual will not necessarily be aware of this.

In normal circumstances, there is no rebound effect and the vasoconstriction lasts for about one hour following the exercise; resistance then returns to normal. In athletes, especially long-distance runners or cyclists, a rebound effect is detectable: after a short period of improved nasal patency, the nose blocks for a considerable length of time, which may affect the sports performance.

When prescribing medication for an athlete, one should consider two principles:

1) the medication should not be on any list of doping products
2) the medication should not adversely affect sports performance

1) The following are regarded as doping:
   a) Vasoconstrictors
      – β-phenylethylamine derivatives
      – ephedrine (oral and nasal)
      – pseudoephedrine (oral and nasal)
      Many combination preparations contain both an antihistamine and a vasoconstrictor
   b) Systemic corticosteroids
   c) Topical steroids – these are allowed if the athlete can submit a declaration from the prescribing physician on the therapeutic indications

2. The following have an influence on physical performance:
   a) First generation antihistamines have a sedating and anticholinergic effect
   b) Immunotherapy may cause discomfort at the site of the subcutaneous injection for several days.

Taking into account these considerations, the ideal treatment for an athlete with allergic rhinitis consists of a second generation H_1-antihistamine and a topical corticosteroid (Van Cauwenberge, 1992). In cases of seasonal rhinitis, immunotherapy can reduce the need or amount of additional medication. This should be started three months before the sports season and the patient is advised against heavy physical exercise on the day of the injection. Athletes should not exercise on the day of injection.

Occupational rhinitis

Occupational diseases are quite common and appear to be increasing. The incidence is probably underestimated because of diagnostic failure and a reluctance of workers to complain in case it jeopardises their jobs.

Occupational exposures have long been recognised as the cause of significant allergic and/or irritant lung and skin disease but it is now realised that the same factors are of equal importance in the nose. Low molecular weight substances found in the working environment were not formerly believed to be of sufficient size to elicit an immunologic response but now appear to be excellent haptens.

Occupational rhinitis may be defined as 'rhinitis caused by exposure to an agent in the workplace'. It often co-exists with occupational asthma. In general, nasal symptoms that follow contact with materials at work appear to parallel the time course of bronchial symptoms and presumably result from the same underlying pathogenesis (Juniper et al., 1993). While the incidence of occupational rhinitis is unknown, a survey of laboratory workers with allergic respiratory symptoms demonstrated that while 100% had rhinitis and conjunctivitis, only 71% had asthma (Lutsky & Neuman, 1975).

Some causative agents for allergic or irritant occupational rhinitis include:
storage mites
psyllium
guar gum
rats
Bacillus subtilis enzymes
western red cedar
isotonic acid hydrazine
trimellitic anhydride (TMA).

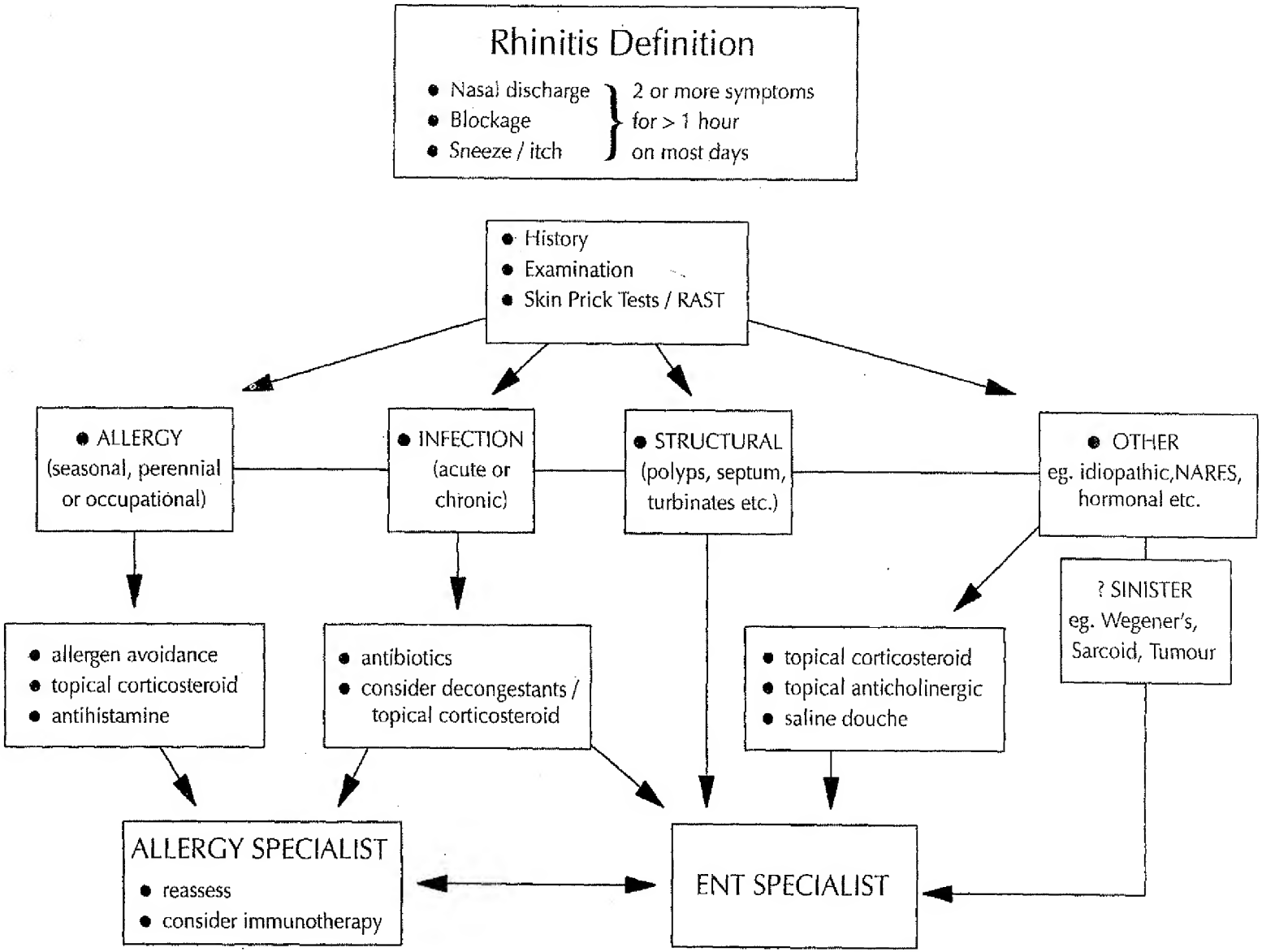
Evaluation and diagnosis of occupational rhinitis require a thorough history and physical examination because occupational rhinitis may be associated with a late-phase reaction, 6–8 hours after exposure to the inciting agent. Whilst symptoms usually abate away from the workplace at weekends and holidays, chronic exposure may produce chronic symptoms unless avoidance lasts a week or longer. Physical examination reveals only non-specific signs of inflammation. Direct nasal challenge can be evaluated by symptom scores or assessment of nasal patency. These tests often require carefully graded exposure to increasing doses of the suspected agent in specialised ventilated environmental exposure chambers. Referral to a chest physician or allergist with a special interest in occupational rhinitis and asthma is generally required if occupational rhinitis is suspected.

Once the diagnosis is established, several approaches are possible. Adequate ventilation in the work environment may be sufficient to reduce exposure to tolerable levels or the use of masks may be appropriate. Finally, conventional pharmacotherapy with antihistamines, topical nasal steroids, sodium cromoglycate and other therapies should be instituted.

Rhinitis medicamentosa

Rebound congestion can result from prolonged intranasal decongestant (α-adrenoceptor agonist) abuse. Therefore, these drugs should not be taken for more than 7–10 days (Proctor & Adams, 1976). Once recognised, the cause of the symptoms should be discussed with the patient. Saline douches and topical nasal steroids are given, whilst the decongestant is gradually stopped. The reason for the original use of the decongestant should be considered. Occasionally, if there are no contraindications, a short course of oral steroids (e.g., prednisolone 30 mg daily for five days) may be helpful.

Rhinitis Management



Stepwise approach to the treatment of rhinitis

SEASONAL ALLERGIC RHINITIS

Allergen avoidance

Mild disease or with occasional symptoms

Rapid onset oral non-sedating H_1-antihistamines when symptomatic
or
Antihistamine or cromoglycate topically to eyes, nose, or both

Moderate disease with prominent nasal symptoms

Topical nasal steroid daily (start early in season)
plus
Antihistamine or cromoglycate topically to eyes

Moderate disease with prominent eye symptoms

Oral non-sedating H_1-antihistamines daily
or
Topical nasal steroid and sodium cromoglycate topically to eyes

If above ineffective

Refer to specialist for further investigations including
* examination of the nose
* allergy tests
* additional pharmacotherapy, e.g., systemic steroids for crisis situations
* possible immunotherapy

PERENNIAL ALLERGIC RHINITIS IN ADULTS

Allergen avoidance

Topical nasal steroids if long-term exposure

Intermittent disease

Oral non-sedating H_1-antihistamines
± occasional use of oral decongestants

PERENNIAL ALLERGIC RHINITIS IN CHILDREN

Allergen and irritant avoidance, e.g., passive exposure to parental cigarette smoke

Topical nasal sodium cromoglycate spray

Oral non-sedating H_1-antihistamine daily

Topical nasal steroid if above ineffective or if long-term exposure

PERENNIAL NON-ALLERGIC RHINITIS

a) With little watery discharge

Avoidance of irritants and advice to stop smoking

Topical nasal steroids (if effective, may be needed long-term)

If treatment ineffective after 1 month
- consider short term systemic steroids
- ± oral decongestants
- refer to specialist

b) With copious watery discharge

Avoidance of irritants and advice to stop smoking

Topical nasal anticholinergic (ipratropium bromide)

REFER TO SPECIALIST

* **occupational rhinitis** – symptoms occurring predominantly on work days

* **nasal polyps** – bilateral chronic nasal congestion with variable sneezing and discharge but with significant olfactory disturbance

* **rhinitis medicamentosa** – persistent rebound congestion as a result of intranasal decongestant abuse

* **malignancy** – continuous nasal congestion, particularly if unilateral, with bloodstained secretion, or both

All recommendations of treatment strategies depend upon local availability of therapeutic agents

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